VOLUME 1

CECIL TEXTBOOK of MEDICINE

19th edition

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TABLE 9–2. FIFTEEN AREAS OF ENDEAVOR FOR PREVENTIVE MEDICINE ESTABLISHED BY THE U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Topics that Are Covered in Chapters of this Section
Smoking and health
Injury prevention
Control of stress and violent behavior
Nutrition
Physical fitness and exercise
Misuse of alcohol and drugs
Immunization
Topics that Are Addressed Elsewhere in this Book
High blood pressure
Sexually transmitted diseases
Toxic agents
Occupational safety and health
Infectious diseases
Topics that Are the Concern of Other Specialties
Family planning
Pregnancy and infant health
Fluoridation and dental health

20 years. The chief component of the decline is coronary heart disease, which has decreased more rapidly in the United States (2 per cent per year) than in any other nation. It seems reasonable to attribute this, at least in part, to the changes in lifestyle that are occurring in this country: the substantial decline in the national prevalence of smoking and of inadequately treated hypertension, the decrease in the mean serum cholesterol level, and the movement to become more physically fit.

The extent and thrust of preventive medicine today have been established by formal health goals in 15 areas of endeavor, created by the U.S. Department of Health and Human Services (Table 9–2). For each of these topics, there are specific objectives for the nation to achieve by the year 2000 that address health status, risk factor levels, public and professional awareness, provision of health services, and mechanisms for evaluation. This section of *Cecil Textbook of Medicine* addresses 7 of these 15 topics that are part of personal health care.

SUMMARY

The emergence of chronic and noninfectious disease as the predominant cause of death and disability in western nations has been accompanied by a growing importance of lifestyle factors as causal agents in health and disease. Among these, cigarette smoking is the single most important modifiable health hazard; abuse of alcohol and other substances, sedentary lifestyle, and improper diet are also important. The clinician's role in preventive medicine still begins with immunization and treatment of such medical conditions as hypertension, but it now extends to health counseling: examining a patient's risk factors, educating the patient, listening to preferences for changing (or not changing) lifestyle, implementing the appropriate behavioral interventions, and following up on these personal health care strategies over the years.

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10 Tobacco and Health

David M. Burns

Cigarette smoking is the largest preventable public health problem in the United States. An estimated 390,000 deaths per year, one sixth of the total mortality in the United States, occur prematurely secondary to the smoking habits of the American population.

Tobacco use, both oral and smoking, was introduced to European settlers by the American Indian, and tobacco was one of the main cash crops in revolutionary America. The invention of a cigarette-making machine in the 1880's and, around the turn of the century, of matches that could be carried safely resulted in a marked shift in tobacco consumption from predominantly pipes, cigars, and chewing tobacco to predominantly cigarettes. Per capita cigarette consumption in the United States increased from 54 in 1900 to a peak of 4336 in 1963. This dramatic switch to cigarette use was followed some 20 to 25 years later by an equally dramatic rise in deaths from lung cancer. The risks associated with tobacco smoking appear to be closely related to the amount of smoke inhaled. Smokers who have used only pipes or cigars tend not to inhale, and therefore the majority of the health risks are correlated with cigarette consumption (Table 10–1).

In the early part of the century, cigarette smoking was largely a male habit, but in the late 1930's and early 1940's women began to smoke in large numbers. Currently, smoking habits in young adults are similar for the two sexes. The prevalence of cigarette smoking is declining in both men and women in the United States population. In contrast, a new marketing effort for smokeless tobacco has led to a major resurgence of snuff use, particularly among adolescent males.

CIGARETTE SMOKE

Tobacco smoke is a complex mixture of some 4000 individual constituents. The smoke is a combination of pyrolysis and distillation products distributed between a particulate phase and a gas phase. Tar, the total particulate matter of the smoke once the water vapor and nicotine have been removed, is the major carcinogen of whole smoke. The gas phase of the smoke has a number of irritating and ciliotoxic agents, as well as high levels of carbon monoxide.

FACTORS DETERMINING RISK

The risks due to cigarette smoking vary with differences in individual smoking habits and with the presence of other risk factors. The risk increases with increasing number of cigarettes

TABLE 10–1. INCREASED RISKS FOR CIGARETTE SMOKERS

Cardiovascular Disease
Coronary artery disease
Peripheral vascular disease
Aortic aneurysm
Stroke
Cancer
Lung
Larynx, oral cavity, esophagus
Bladder, kidney
Pancreas
Lung Disorders
Cancer (as noted above)
Chronic bronchitis with airflow obstruction
Emphysema
Complications of Pregnancy
Infants—small for gestational age, higher perinatal mortality
Maternal complications—placenta previa, abruptio placentae
Gastrointestinal Complications
Peptic ulcer
Esophageal reflux
Other
Altered drug metabolism

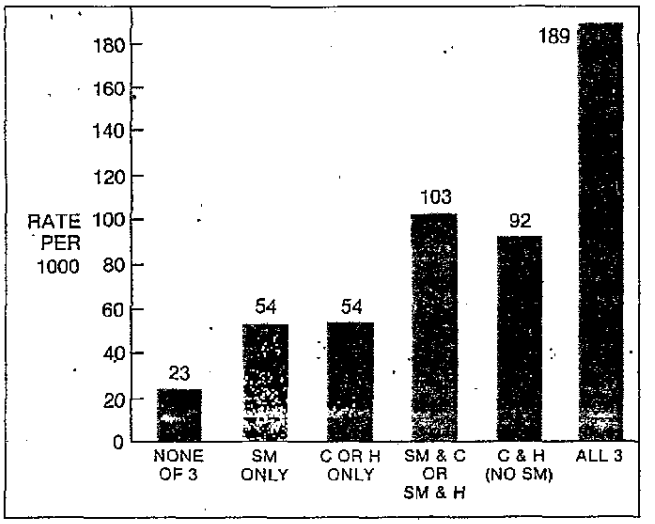

FIGURE 10–1. Major risk factor combinations, 10-year incidence of first major coronary events, men age 30 to 59 at entry, Pooling Project. Risk factor status at entry: Definitions of the three major risk factors and their symbols are hypercholesterolemia (C) = ≥ 250 mg/dl; elevated blood pressure (H) = diastolic pressure ≥ 90 mm Hg; cigarette smoking (SM) = any current use of cigarettes at entry.

smoked per day, depth of inhalation, and duration of the smoking habit. The risk also increases with the younger age at which regular smoking is begun.

A given dose of smoke exposure may interact with other personal characteristics or environmental exposures to magnify the risk of disease greatly. Thus, the risks incurred by cigarette smoking in someone with elevated blood pressure or high levels of asbestos exposure are much larger than the risks for smokers without those characteristics. In addition, the presence of smoking-induced disease in one organ system (e.g., chronic obstructive lung disease) may alter the ability to treat or survive a second disease process (e.g., lung cancer).

CARDIOVASCULAR DISEASE

Cigarette smokers have almost twice the risk of nonsmokers of developing a myocardial infarction or dying of coronary heart disease. This relative risk of heart disease is even greater at younger ages, when the incidence of disease would otherwise be very low. The relative risks for sudden death from coronary disease, peripheral vascular disease, and aneurysm of the aorta are even higher. In contrast, cigarette smokers have only a slightly greater risk of developing angina pectoris.

The magnitude of the risk of coronary heart disease associated with cigarette smoking is equivalent to the risks associated with elevated blood pressure or elevated serum cholesterol. The per cent of the population with smoking as a risk factor is substantially larger than the percentage with either elevated blood pressure or elevated serum cholesterol. As a result, *smoking ranks as the largest avoidable cause of coronary heart disease in the American population.*

Cigarette smoking acts as an independent risk factor for coronary heart disease; that is, its effect is not explained by levels of other risk factors. When more than one risk factor is present, however, smoking interacts with the other major risk factors to increase the risk synergistically (Fig. 10–1). The presence of smoking, or of either of the other risk factors, increases the risk by 31 per 1000, compared with the risk of someone with none of the risk factors. The presence of a second risk factor in someone who smokes results in an increase in risk of 49 per 1000 over the risk when only one risk factor is present, and the addition of a third risk factor increases the risk by 86 per 1000. The actual risk is always greater than the sum of the risks measured independently, suggesting that when multiple risk factors are present, they interact to create more disease. This interaction may occur by accelerating the development of atherosclerosis or by increasing the likelihood or severity of a myocardial infarction for any given level of atherosclerosis.

Smokers have more atherosclerosis than nonsmokers, particularly in the aorta. Smoking a cigarette raises heart rate and blood pressure, necessitating a greater myocardial oxygen delivery, while the carbon monoxide in the smoke increases the blood's carboxyhemoglobin level, thus decreasing its oxygen-carrying capacity. Cigarette smoking also increases platelet adhesiveness and lowers the threshold for ventricular fibrillation and may thereby play a role in the acute events surrounding some thrombotic myocardial infarctions.

Cigarette smoking has a more profound effect on the peripheral vascular bed than on the coronary or cerebral vessels. Over 90 per cent of patients with atherosclerotic peripheral vascular disease are cigarette smokers. Cessation of cigarette smoking is critical to treatment of these patients. In those who fail to quit, there is a higher incidence of amputation, and surgical therapy is dramatically less successful.

The risk of coronary heart disease due to smoking is present at all ages beyond 30, but smoking is responsible for a greater proportion of coronary deaths in younger age groups than in older age groups. This risk declines dramatically with the cessation of cigarette smoking. By 5 years after the last cigarette, the risk in those who had smoked less than one pack per day approximates the risk in lifelong nonsmokers. For those who had smoked more than one pack per day, a small residual risk of coronary heart disease may persist.

CANCER

Lung cancer is the largest cause of death from cancer in men and women (Ch. 68). *Approximately 85 per cent of mortality due to lung cancer is causally attributed to cigarette smoking and is therefore potentially preventable.*

Cigarette smokers are ten times more likely to develop lung cancer than nonsmokers. This risk is proportional to the number of cigarettes smoked per day, increasing to 20 to 25 times the risk of the nonsmoker in those who smoke two or more packs of cigarettes per day. The risk is also increased in those who inhale more deeply or began smoking at a younger age. Lung cancer death rates begin to increase rapidly after age 35 (Fig. 10–2). Cigarette smoking causes all of the major types of lung cancer, including squamous cell, adenocarcinoma, oat cell, and large cell

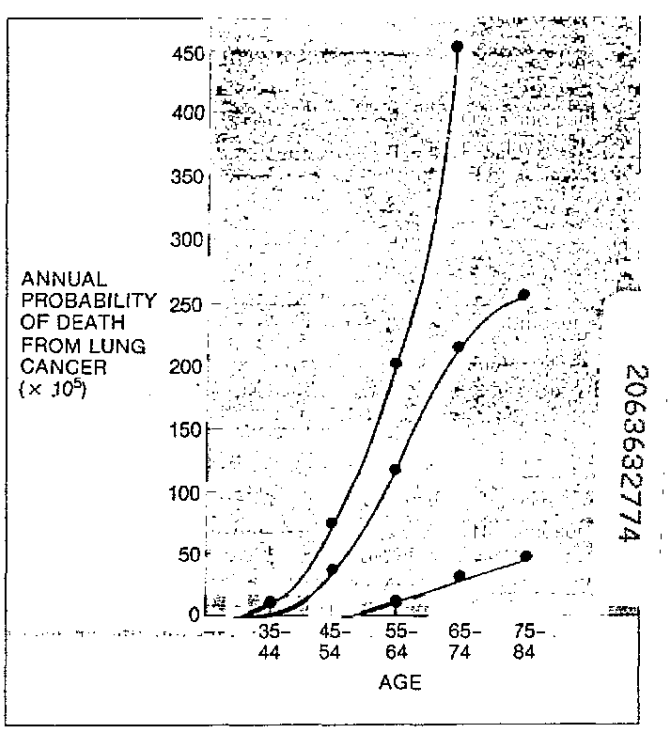

FIGURE 10–2. Annual death rate from lung cancer in nonsmokers, smokers in general, and those who smoke more than one pack per day.

carcinoma. Asbestos exposure and uranium mining interact with cigarette smoking to increase the risk of lung cancer dramatically.

The relative risks of developing *laryngeal cancer* for the cigarette smoker closely track those of lung cancer, but the total number of cases is smaller and the survival better. Cigarette smokers are five times more likely to develop *cancer of the oral cavity and esophagus*, and there appears to be a synergistic interaction between cigarette smoking and alcohol consumption for cancer of the larynx, oral cavity, and esophagus. Cigarette smoking is also a major contributing factor in *cancers of the bladder, kidney, and pancreas*, and an association between cigarette smoking and *gastric and cervical cancers* has been noted. The use of chewing tobacco or snuff can cause cancers of the cheek or gum. Overall, tobacco consumption is responsible for approximately 30 per cent of the total United States cancer mortality.

Cigarette smoking induces changes in the respiratory epithelium that progress from hyperplasia to dysplasia and even to carcinoma in situ. Tobacco smoke contains a variety of tumorigenic agents, including several that can act as complete carcinogens. The impact of these tumorigenic agents may be magnified by the ciliotoxic agents in the smoke that interfere with the normal clearance mechanisms of the lung and result in a prolonged retention of the carcinogenic agents in the lung.

Cessation of cigarette smoking results in a lessening of the risk of cancer in comparison with the risk to the continuing smoker. The risk for light smokers approximates the risk of the nonsmoker by 10 to 15 years after cessation. Heavy smokers have a residual two- to threefold increased risk that is proportional to their lifetime exposure to smoke.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Cigarette-induced lung injury is characterized by three overlapping syndromes: cough and mucus hypersecretion, bronchitis with airflow obstruction, and emphysema (see Ch. 58). By age 60 most cigarette smokers have changes in the airways and some degree of pathologic emphysema, but only the minority have symptomatic ventilatory limitation. The prevalence of cough increases in cigarette smokers by the early teens, and the small airways are abnormal in many smokers by early adulthood. Whether either of these changes predicts those who will eventually go on to develop symptomatic chronic airflow limitation is unclear.

The cigarette smoking habit is the major predictor in a population for the development of COPD. The prevalence of COPD and risk of death from COPD increase with the number of cigarettes smoked per day and the depth of inhalation, as does the prevalence of chronic cough and sputum production, rate of decline in the measurements of expiratory airflow, and degree of anatomic emphysema.

In contrast to nonsmokers, the majority of cigarette smokers examined at autopsy have some degree of emphysema and hypertrophic changes of the respiratory epithelium. However, only a minority of cigarette smokers manifest clinically significant airflow obstruction. Those who develop chronic airflow obstruction may be a subset of the smoking population identifiable by a rapidly declining FEV_1 early in the course of disease. In any event, it is rare for symptomatic chronic airflow obstruction to develop in anyone who maintained normal measures of expiratory airflow through age 45.

Cessation of cigarette smoking is of some benefit at all preterminal stages of ventilatory impairment. Changes in the small airways and early declines of FEF_{25-75} may reverse within one year of cessation. Cough and sputum production also lessen, and the annual rate of decline in measures of expiratory airflow moderates and approximates the rate of decline in nonsmokers. These changes are probably related to reversal of the chronic inflammatory changes in the large and small airways and the recovery of ciliary function, as there is no evidence that the emphysematous process is reversible.

Lungs of smokers contain increased numbers of alveolar macrophages and polymorphonuclear leukocytes, probably drawn there as part of the inflammatory response to the irritants in the smoke. These cells release elastase, which is capable of degrading the structural elements of the lung, resulting in a loss of elastic recoil. This destructive process is normally limited by bloodborne antiproteases. However, cigarette smoke contains a number of oxidants that destroy the function of these protective proteins, and the result is an imbalance in the protease-antiprotease system favoring degradation and rupture of alveolar walls.

RISKS FOR WOMEN

Being female does not protect against the risks of developing cancer or chronic lung disease. Much of the premenopausal difference in cardiovascular risk enjoyed by women disappears in those who smoke.

In addition to the risks defined for men, women also incur additional risks related to pregnancy and use of oral contraceptives. Infants of smoking mothers are small for their gestational age in weight, length, and head circumference, and they experience a higher perinatal mortality, particularly if other determinants of a high-risk pregnancy are present. The smoking mothers are also at greater risk for the maternal complications of pregnancy, especially placenta previa and abruptio placentae.

Women who smoke and use oral contraceptives greatly increase their risk of cardiovascular disease. They are over 30 times more likely to develop a myocardial infarction, and about 20 times more likely to have a subarachnoid hemorrhage, than their nonsmoking peers who do not use oral contraceptives.

INVOLUNTARY SMOKING

Environmental tobacco smoke contains most of the toxic and carcinogenic compounds identified in mainstream smoke; and therefore the question is not whether these agents can cause disease, but rather whether the dose and mode of exposure experienced in involuntary smoking carry a measurable risk. Absorption of smoke constituents from the environment has been documented in both infants and adults, and a number of epidemiologic studies have demonstrated health effects in humans.

Involuntary smoking can cause lung cancer in nonsmokers. The risk is small in comparison to active smoking but is large in comparison to other carcinogenic exposures experienced by the general population. From 500 to 5000 lung cancers per year have been estimated to result from involuntary smoking.

The majority of nonsmokers express annoyance and experience eye and respiratory tract irritation on exposure to smoke. Individuals with pre-existing disease may become more symptomatic on exposure to smoke, particularly those with allergies, and possibly those with chronic heart and lung disease. Nonsmokers with long-term exposure to environmental tobacco smoke may develop changes in the small airways of the lung.

Infants of smoking parents have a higher incidence of bronchitis and pneumonia in the first year of life, and the children of smoking mothers experience a developmental lag in lung growth.

CIGARETTES WITH LOW TAR AND NICOTINE

The machine-measured yield of tar and nicotine for the average cigarette smoked by the American population has been steadily declining. Smokers of lower yield cigarettes have a slightly lower risk of lung cancer than smokers of the high-yield cigarette, but this benefit disappears if they increase the number of cigarettes they smoke per day. There is also a lower prevalence of cough and phlegm, but probably no major impact on the risk of developing cardiovascular disease or chronic airflow obstruction. There are two major reasons why the decline in machine-measured tar and nicotine yield has not been accompanied by a concomitant reduction in biologic effect: (1) Many smokers may compensate for the decline in yield by increasing the number of cigarettes smoked per day, or by inhaling more deeply, thereby negating any possible reduction in smoke exposure "dose." (2) The machine-measured yield may not correspond to the yield when the cigarette is actually smoked. This is particularly true for the very low-yield cigarettes that have vents or channels designed into the filter so that the machine draws very little smoke through the filter. These vents can be occluded by the smoker, or the volume of the puff increased, with a resultant dramatic rise in the yield. For these cigarettes, the measured tar and nicotine yields have almost no relation to either actual yield or biologic potency.

An additional concern is the wide variety of flavoring and other additives that have been used to compensate for the decline in tobacco content. These additives are considered trade secrets and may be added to the cigarette without informing the public of their presence and without any review for toxic effects. These additives represent a major gap in the understanding of the disease risks associated with smoking the modern cigarette.

OTHER EFFECTS

Cigarette smokers have a greater incidence of gastric and duodenal ulcers and delayed healing of these ulcers. Smoking also relaxes the esophageal sphincter and may contribute to esophageal reflux.

Several of the constituents of tobacco smoke are capable of inducing hepatic microsomal systems, which then alter the metabolism of other drugs. Theophylline, phenacetin, antipyrine, caffeine, and imipramine are metabolized more rapidly by smokers, and adjustment in the dosage may be required with cessation. Smokers have lower blood levels of vitamins C and B_{12}. Hematocrit and hemoglobin levels, as well as carboxyhemoglobin levels, are elevated in smokers; and smoking is one cause of an elevated red cell volume. Smokers also have small alterations in the other diagnostic tests, including a higher leukocyte count, but these differences are not usually clinically significant for an individual patient.

Pipe and cigar smokers who have never smoked cigarettes have a lower risk of cardiovascular disease, lung cancer, and chronic airflow obstruction than do cigarette smokers. They have similar risks of cancer of the upper respiratory tract. These differences are due to the tendency of pipe and cigar smokers not to inhale the more irritating smoke of these forms of tobacco. Cigarette smokers who switch to pipes and cigars do tend to inhale, however, and so it is not clear that switching to a pipe or cigars results in a lowering of the risks for the cigarette smoker.

The re-emergence of oral snuff use among male adolescents in the last several years has generated substantial public health concern. Smokeless tobacco use can cause cancer of the cheek and gum and gingival recession. It may also increase the risk of other oral cancers, and regular use of snuff can lead to nicotine addiction.

SMOKING BEHAVIOR AND CESSATION

Regular cigarette smoking begins almost exclusively during adolescence; 90 per cent of smokers begin before age 20. The availability and relatively low cost of cigarettes coupled with peer pressure and the desire to model adult behavior are determinants of adolescent smoking. Tobacco advertising may also influence the initiation of regular smoking by creating an image of the smoker as a secure, confident, successful, in control, and attractive individual. By smoking, adolescents are able to superimpose this positive image created by advertising on their own inadequate self-image and thereby feel better. Those adolescents with the least external validation of their self worth (through academic, athletic, or social achievements) are the ones most in need of manipulation of their internal self-image and, correspondingly, most susceptible to the images presented by advertising.

Addiction to cigarettes depends on nicotine. Beyond the pharmacologic stimulus of nicotine, however, the smoker usually creates a series of learned responses that reduce stress and alter mood. The pattern of tobacco use therefore merges into the way that the smoker learns to deal with the world. Cessation of smoking requires that the smoker give up a major coping mechanism.

Smoking cessation clinics have long-term success in 30 to 40 per cent of the smokers who persevere in their programs, but comparatively few smokers are willing to participate in these clinics. Current tobacco control strategies emphasize altering the environment in which the smoker lives by making smoking socially unacceptable, by increasing the cost of cigarettes, and by limiting the locations in which it is permissible to smoke.

Physicians can effect sustained cessation of smoking in a substantial number of patients if they are willing to treat smoking as a potentially serious medical problem. This requires obtaining information, defining a therapeutic plan, and following the results of that therapy. The information to be obtained includes the smoking status, a history of past cessation attempts and the methods used, as well as the current interest in quitting. In addition, the time from awakening to first cigarette is a measure of the strength of the addiction and may be useful in deciding whether to prescribe pharmacologic aids to the cessation attempt. In their offices physicians can ask the patient to quit, can motivate the attempt, and can negotiate a date for quitting. No smoking patient should leave the office without understanding that his or her smoking is a major health problem. The responsibility of the physician is not to get all patients to quit on a single visit, but to move each smoking patient closer to cessation on each visit. Those who have not thought about quitting should think about it; those who are thinking about it should try; and those who have tried and failed should be motivated to try again. Smokers should be encouraged to quit "cold turkey" rather than tapering down. The follow-up of cessation advice is also critical, not only because it reinforces the importance of cessation for the patient, but also because it improves the likelihood of successful cessation. A simple letter of encouragement from the physician 2 weeks following the quit date may substantially improve patient motivation and success.

The use of nicotine gum increases the chance of successful short-term cessation when utilized with an appropriate behavioral intervention program. Clonidine, particularly when used as a patch, has shown promise as a means of reducing the withdrawal symptoms but remains an investigational drug for this purpose.

Effective smoking intervention by the physician can be delivered in 3 to 5 minutes using the above approach. Physicians should refer patients who need more extensive assistance to programs designed to provide this assistance. A variety of community organizations provide cessation assistance, both in groups and as self-help materials, and these organizations can be located in the telephone directory or by contacting the local heart, lung, or cancer societies.

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11 Control of Unintended Injuries and Those Due to Violence

Stephen B. Hulley

Deaths from injury are the fourth most common cause of death in the United States; they number more than 150,000 each year and are the leading cause of death for young and middle-aged people in the age range 1 to 45. The problem is even larger if *nonfatal* injuries, some of which cause permanent disability, are considered: There are several hundred injury-related emergency room visits for every death from injury. One third of all injury